November 1, 1991, Volume 199, Number 9

JOURNAL

OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

JOURNAL

OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

© American Veterinary Medical Association, 1991
All Rights Reserved

Vol 199 November 1, 1991 No. 9
JAVMA 199 (9) 1113-1232 (1991)
ISSN 0003-1488

Interpretive Summaries of Articles in This Issue ... 1116

Letters

Evaluating copper and zinc in dairy rations ... 1119
Serologically atypical canine ehrlichiosis associated with *Ehrlichia risticii* "infection" ... 1120
Veterinary history ... 1120
Preparing for disasters ... 1121

News

AVMA at Work—Winds of change blow strong at public corporate practice symposium ... 1123
NBEC approves plans for new format and scoring system ... 1126
Hitting the campaign trail ... 1128
Awards and Honors—Harvey receives Salmon Award ... 1128
Insurance Information—Trust examines insurance consideration for animal-allergic veterinarians ... 1129
Washington Watch—Crawford resigns as FSIS administrator ... 1130
Under the Umbrella—AAHA to build new headquarters ... 1130
Convention Spotlight—Computer program on-line at Seattle convention ... 1131
Regulatory Front—List of restricted-use pesticides mounts ... 1132
Out and About ... 1136
Student and School Scenes—Dean appointed at Atlantic Veterinary College ... 1137
Coast to Coast ... 1138
Deaths ... 1140

New Feature

Timely Topics in Nutrition

Dietary fiber in small animal therapeutics
Donna S. Dimski and C. A. Buffington ... 1142

Reflections . . .

How to keep your sanity, self-respect, and dignity in a rural practice—*Al Doughty* ... 1147

Legal Brief

Wills requiring the destruction of pets—the veterinarian's position—*Harold W. Hannah* ... 1156

Reports of Original Studies

In situ observation of the ovaries of llamas and alpacas by use of a laparoscopic technique
Julio Sumar and P. Walter Bravo ... 1159

Evaluation of intra-abdominal vasectomy in llamas and alpacas—*P. Walter Bravo and Julio Sumar* ... 1164

Effect of storage on measurement of ionized calcium and acid-base variables in equine, bovine, ovine, and canine venous blood
Otto Szenci, Endre Brydl, and Csaba A. Bajcsy ... 1167

Clinical Reports

Escherichia coli bacteremia associated with hypertrophic osteodystrophy in a dog
Kurt S. Schulz, John T. Payne, and Everett Aronson ... 1170

Bilateral wedge ostectomy on an 18-month-old llama with severe bilateral carpal valgus
K. R. E. Squire and S. B. Adams ... 1174

Lymphosarcoma with urinary bladder infiltration in a horse—*Raymond W. Sweeney, Amir N. Hamir, and Robert R. Fisher* ... 1177

Rostral mandibulectomy in five horses
D. W. Richardson, L. H. Evans, and E. P. Tulleners ... 1179

Pharmacologic treatment of priapism in two horses—*Deborah V. Wilson, Frank A. Nickels, and M. Amy Williams* ... 1183

Reports of Retrospective Studies

Clinical management and surgical repair of atresia coli in calves: 66 cases (1977-1988)—*Donald F. Smith, Norm G. Ducharme, Susan L. Fubini, William J. Donawick, and Hollis N. Erb* ... 1185

Clinical, clinicopathologic, and pathologic features of plague in cats: 119 cases (1977-1988)—*Millicent Eidson, John P. Thilsted, and Ole J. Rollag* 1191

What Is Your Diagnosis?

Jacek J. de Haan, Gary W. Ellison, and Norman Ackerman 1199

Book Reviews

Radiology and Ultrasound of Urogenital Diseases in Dogs and Cats 1163
Swine Nutrition Guide 1197

Announcements

Coming Meetings 1201
Foreign Meetings 1204
Certification Examinations 1207
State Board Examinations 1207
Applications for Membership 1208
Educational Opportunities 1209

Classified Ads 1216

About the cover 1124

INSTRUCTIONS TO AUTHORS 1198

The AVMA headquarters new address and telephone and facsimile numbers are:
American Veterinary Medical Association
1931 N. Meacham Rd.
Suite 100
Schaumburg, IL 60173-4360
Telephone: 708/925-8070
Telephone toll free: 1-800/248-2862
Fax: 708-925-1329

JAVMA STAFF

Dr. Albert J. Koltveit, *Editor-in-Chief*
Dr. Janis H. Audin, *Associate Editor*
Dr. Nancy W. Leveque, *Assistant Editor*
Dr. Kathleen J. Kovacs, *Assistant Editor*
Dr. Leighann Daristotle, *Assistant Editor*
Dr. Candace S. Lundin, *Assistant Editor*
Dr. Richard L. Torbeck, *Assistant Editor*
Bernadine Gordon Clune, *Production Manager*
Susan Kahler, *Senior News Editor*
Lea Spencer, *Associate News Editor*
Paul Zuziak, *Assistant News Editor*
Irene Jansons, *Advertising Production Manager*
Jerry Schwartz, *Production Coordinator*
Kim Luenzmann, *Production Coordinator*
Pete Johlie, *Editorial Assistant*
Linda Behzad, *Editorial Assistant*
Kimberly Schmitt, *Editorial Assistant*
Clinton Ohlers, *Editorial Assistant*
Jody Hundley, *Editorial Assistant*
Opal Anderson, *Advertising Assistant*
Liane Lenski, *Librarian*

ADVERTISING SALES

East—C. Lynn Coy & Associates
PO Box 15260, Park Square Station
Stamford, CT 06901, 203/327-4626
Midwest—Lampert & Associates
PO Box 325, Western Springs, IL 60558
708/482-3993
West—Lane E. Weiss & Associates
582 Market St, Suite 611
San Francisco, CA 94104
415/986-6341

1991-1992 AVMA OFFICERS

Dr. Gerald L. Johnson, *President*
Dr. L. Everett Macomber, *President-Elect*
Dr. M.T. Szatalowicz, *Vice President*
Dr. Owen R. Stevens, Jr., *Treasurer*
Dr. Sherbyn W. Ostrich, *Executive Board Chairman*
Dr. A. Roland Dommert, *Executive Vice President*
Dr. Bruce W. Little, *Assistant Executive Vice President*

AVMA SCHAUMBURG OFFICE

American Veterinary Medical Association, 1931 N Meacham Rd, Suite 100, Schaumburg, IL 60173-4360. Office hours: 8:30 am to 4:45 pm, Monday through Friday. Telephone toll free: 1-800/248-AVMA. Outside the continental United States call 708/925-8070. Fax: 708/925-1329.

AVMA GOVERNMENTAL RELATIONS DIVISION

Dr. William R. Van Dresser, 1023 Fifteenth Street, NW, Suite 300, Washington, DC 20005-2602. Telephone toll free: 1-800/321-1473. Outside the continental United States call 202/659-2040. Fax: 202/842-4360.

SUBSCRIPTIONS: For one year: members $35; others $70 in the United States and possessions; $90 in foreign countries. Single copies: $6.00 in the United States and possessions; $8.00 in foreign countries.

Published semimonthly at 1931 N Meacham Rd, Suite 100, Schaumburg, IL 60173-4360, by the American Veterinary Medical Association. Second class postage paid at Schaumburg, Ill, and at additional mailing offices. Contents copyright 1991 by the AVMA.

ADDRESS CHANGE: Send new address (and moving date) 45 days ahead. Include old address as it appeared on previous issues (if possible send label).

POSTMASTER: Send address changes to JAVMA, 1931 N Meacham Rd, Suite 100, Schaumburg, IL 60173-4360.

VBPA

Clinical, clinicopathologic, and pathologic features of plague in cats: 119 cases (1977–1988)

Millicent Eidson, MA, DVM; John P. Thilsted, DVM, PhD; Ole J. Rollag, DVM

Summary: *The clinical, clinicopathologic, and pathologic features of 119 cases of plague in cats from 1977 to 1988 in New Mexico were reviewed. Fifty-three percent were bubonic, 10% were pneumonic, 8% were septicemic, and 29% with neither buboes nor pneumonia were unclassified (but presumed septicemic). Three quarters of the lymphadenopathy was submandibular, and almost half of this was bilateral. One third of all cats had the triad of lethargy, anorexia, and fever in addition to buboes; one quarter had this triad plus abscesses. The overall mortality rate was 33%, with the greatest risk of death in pneumonic cases. For confirmatory diagnosis with a single laboratory test, fluorescent antibody was most frequently used (39% of cases). Cultures and passive hemagluttination titers were also used for confirmation. Gross and histologic findings depended on the type of plague with* Yersinia pestis *organisms visualized in buboes of cats with bubonic plague and in the alveolar spaces and respiratory tubules of cats with pneumonic plague.*

Plague, caused by the bacteria *Yersinia pestis*, is a disease of antiquity, which has decimated the world's human population at various stages in history. Plague continues to be an important disease in many parts of the world today, including the western United States. Most infections of rodents, lagomorphs, human beings, wild and domestic canids and felids, and other animals result from the bites of infected fleas. However, transmission can occur through any blood contact during handling, skinning, or eating infected animals.

Although severe morbidity and mortality for human beings has been well documented for centuries, plague's effects on the health of domestic and companion animals has only recently begun to be understood. It now appears that plague in cats is an important disease impacting the health of cats and their owners as well as veterinary staff who come in contact with them. Sporadic feline plague cases have been reported in the United States and elsewhere.[1-13] Transmission of plague to human beings via the bites of rodent fleas on cats, direct transmission of feline infection through cat bites, or contact with draining abscesses or pneumonic aerosals has been documented in at least 15 cases.[1-5, 7-17] The risk factors for cats acquiring plague infection in New Mexico have been recently reviewed and appear to include hunting rodents, particularly in the summer, in rural areas.[14] The purpose of the study reported here was to determine the clinical, clinicopathologic, and pathologic features of plague in cats.

Criteria for Selection of Cases

After the first laboratory-confirmed plague case in a cat was reported in New Mexico in September 1977, a registry of cases was established by the Office of Epidemiology, New Mexico Department of Health (DOH). Laboratory confirmation for suspect cases was provided by DOH's Scientific Laboratory Division and the New Mexico Department of Agriculture's Veterinary Diagnostic Services. Plague confirmation is not available at any other laboratories in New Mexico, and there were no reports of veterinarians who use out-of-state laboratories for plague testing.

Cases were confirmed with isolation of *Y pestis* from culture material, positive fluorescent antibody (FA) stain from clinical or pathologic material, a

From the Office of Epidemiology, New Mexico Department of Health, PO Box 26110, Santa Fe, NM 87502 (Eidson, Rollag) and Veterinary Diagnostic Services, New Mexico Department of Agriculture, 700 Camino de Salud NE, Albuquerque, NM 87106 (Thilsted).

We thank the following for providing information essential for completing this study: L. Nims, K. Montman, and Drs. M.H. Hume, J.R. Jackson, M. Johnson, K. Blackshear, R. Bouloy, F.H. Coons, R.J. Cortesi, F.K. Sweeney, M.A. Hedberg, T.W. Schuker, C.J. Mabray, and E.T. McKenna, Albuquerque, NM; P. Reynolds, T. Brown, and Drs. J.L. Lauer, R.A. Snook, T.E. Smith, W.J. Arrighetti, P. McCreight, P.J. Hinko, D.W. Krogdahl, B.E. Swanson, S.P. Brandt, R.L. Peterson, and T. Gallegos, Santa Fe, NM; Drs. T.A. Schupbach and W. Wiatt, Taos, NM; Drs. P.L. Curtis and N.E. Hauer, Los Alamos, NM; Dr. D.W. Dessauer, White Rock, NM; Drs. R. Huechen and D. Despres, Cedar Crest, NM; Dr. S.J. Dowds, Tucumcari, NM; Drs. J.F. Wisniewski and B.R. Bowker, Edgewood, NM; Dr. B.G. Thornton, Grants, NM; Dr. F.L. Willard, Ruidoso, NM; Dr. I.R. Sawyer, Raton, NM; Dr. K. Ramsey, Espanola, NM; Dr. K. Podkonjak, Gallup, NM; Drs. E. Staley and E.R. Leslie, Alamogordo, NM; Dr. T.K. Dienlin, Socorro, NM; Dr. A. DeGroot, Sandia Park, NM; and Dr. L. Locklar, Silver City, NM.

fourfold increase in passive hemagglutination titer between acute and convalescent sera, or a single titer of 1:32 or greater with clinical signs compatible with plague infection.

Cases were defined as bubonic if the cat had 1 or more enlarged lymph nodes at clinical or pathologic examination. Because of the substantially increased risk of transmission, pneumonic plague was diagnosed with any clinical, histologic, or radiologic evidence of pneumonia, regardless of whether the lung infection appeared to be primary or attributable to secondary spread. In people with plague, cases of those people without buboes or pneumonia are defined as septicemic. For this study, cases in cats without buboes or pneumonia and without evidence of septicemia were defined as unclassified because feline cases are sometimes not investigated as thoroughly as human cases. Cases were defined as septicemic if the cats were ill and bacteremic as documented with cultures of blood.

Clinical information was obtained from veterinarians and cat owners. Necropsies were performed on available cats.

Results

Signalment—Plague has been reported in 119 domestic cats in New Mexico between September 1977 and Dec 31, 1988 (Fig 1). Affected cats ranged in age from newborn to 15 years, with a mean of 3 years. Sixty-six were males (55%), 38 were females (32%), and gender was unreported in 15 cases (13%). Forty-one percent (49 cats) were reported to be neutered, compared with 16% unneutered (19 cats). Almost half of the cats were reported to be domestic shorthairs (45%, 53 cats). One fifth were domestic longhairs (24 cats), 8 cats were Siamese or Siamese crosses, 5 cats were Manxes, and 3 cats were other breeds. Feline plague cases were distributed throughout most of the state at elevations between 3,500 and 8,600 ft, in 18 of 32 counties, but primarily in the 7-county north-central area (103 cases, 87%).

Clinical findings—The majority of cases (63, 53%) were bubonic without evidence of septicemia or pneumonia. Twelve cases (10%) were pneumonic. Four of these were secondary to bubonic infection, bringing the number of cases with enlarged buboes to 67. The remaining 8 pneumonic cases had no obvious primary focus.

Five of the 12 pneumonic cases were diagnosed clinically by veterinarians, diagnosis of 3 cases was based on the veterinarians' interpretation of radiographs, and diagnosis of 4 cases was based on the identification of *Y pestis* organisms in the alveoli on histologic analysis. Nine cases (8%) with neither buboes nor pneumonia were confirmed to be septicemic with cultures of blood. An additional 35 cases (29%) without buboes or pneumonia did not have cultures of blood submitted to confirm septicemia.

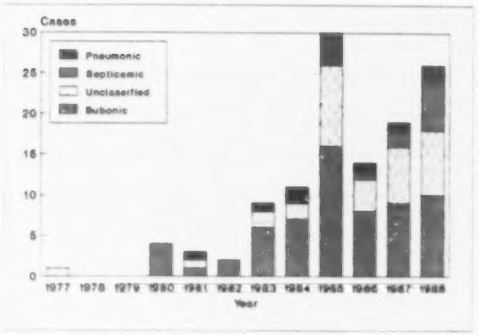

Figure 1—Reported cases of plague in cats in New Mexico from 1977 to 1988 (n = 119).

Of the 67 plague cats with enlarged lymph nodes, 50 (75%) had submandibular lymphadenopathy (Table 1). Nine cats had multiple types of lymph nodes enlarged. Submandibular and cervical buboes were reported in 4 cats, and popliteal and external iliac node enlargement was reported in 2 cats. One cat had enlarged cervical, axillary, and external iliac lymph nodes; 1 cat had enlarged submandibular, prescapular, and popliteal lymph nodes; and 1 cat had enlarged submandibular and external iliac lymph nodes.

Table 1—Lymph node involvement in cats with bubonic plague (n = 67).

Nodes	No. of cases	Bilateral	Right	Left	Unspecified
Submandibular	50	23	5	14	8
Cervical	8	2	0	3	3
External iliac	6	1	1	3	1
Popliteal	6	0	3	2	1
Prescapular	5	0	1	2	2
Axillary	2	0	0	1	1
Unspecified	1	0	0	0	1
Total	78	26 (33%)	10 (13%)	25 (32%)	17 (22%)

Approximately one third of the lymph node enlargement was bilateral, and another one third was on the left side. Right-sided enlargement was reported in only 13% of the cases, although the location of the enlargement was not specified in almost one quarter of the cases. Bilateral involvement was most frequently noticed when submandibular lymph nodes were involved. The other lymph nodes in which bilateral enlargement was noticed were cervical and external iliac.

The size of the lymph node enlargement was reported for 18 cases; ranging from 2.5 to 7.6 cm, with a mean value of 3.8 cm. In 6 cases, the lymph nodes were reported as draining prior to veterinary treatment, compared with no drainage in 32 cases.

Lethargy was reported in 97 cats (82%), anorexia in 92 cats (77%), fever in 85 cats (71%), abscesses in 41 cats (34%), dyspnea in 17 cats (14%), discharge from the mouth or nose in 17 cats

(14%), and coughing or sneezing in 11 cats (9%). The triad of lethargy, anorexia, and fever was reported along with buboes in 40 cats (34%), and along with abscesses in 28 cats (24%). Most of the abscesses were in affected lymph nodes (25 of 41, 61%). Other abscesses were sublingual (5 cases), tongue (1), mouth (2), face (2), lips (1), maxilla (1), gluteal muscle (1), hind limb (1), forelimb (1), fore- and hind limbs (1), thorax (1), and abdomen (1).

Specific temperatures > 39.2 C were recorded in 81 cats. These higher than normal temperatures averaged 40.6 C. Miscellaneous signs reported included (each for only 1 cat): ocular discharge, vomiting, diarrhea, dehydration, coma, ataxia, necrotic stomatitis, facil ulcers, cellulitis, enlarged tonsil, partial alopecia, poor hair coat, massive weight loss, and crepitous body; in addition to oral ulcers (2 cases), swollen tongue (2 cases), and abortions (2 cases).

Survival status varied in relation to the type of plague and antibiotic treatment (Table 2). Although only 10% of the feline plague cases were pneumonic (12 of 119), 21% of the deaths or euthanasias were pneumonic (8 of 39). Septicemic or unclassified (presumably septicemic) cases represented 37% (44 of 119) of the total, compared with 38% (15 of 39) of the deaths. Fifty-three percent of the feline cases were bubonic, but only 41% (16 of 39) of the deaths were bubonic.

Table 2—Survival of cats with plague by type of plague and use of antibiotic treatment (n = 119)

Treated/type plague	Survived	Died	Euthanatized	Total
Treated with antibiotics				
Bubonic	41	1	4	46
Septicemic or Unclassified	26	0	2	28
Pneumonic	3	0	0	3
Total	70	1	6	77
No antibiotic treatment reported				
Bubonic	6	1	10	17
Septicemic or Unclassified	3	7	6	16
Pneumonic	1	5	3	9
Total	10	13	19	42

Most of the 77 cats treated with antibiotics survived, in contrast to the 42 cats not treated with antibiotics. Only one quarter of those survived, with approximately one third dying and almost half euthanatized.

Tetracycline, doxycycline, or vibramycin was used in 43% of those cases for which antibiotics were used, ampicillin, amoxicillin, or hetacillin was used in 16%, penicillin was used in 9%, trimethoprim-sulfadiazine was used in 5%, trimethoprim-sulfamethoxazole was used in 3%, gentamicin was used in 4%, streptomycin was used in 3%, lincomycin was used in 3%, and chloramphenicol was used in 3% of cases. Antibiotic doses, reported in 34 cases, varied widely. For tetracycline, the total daily dose ranged from 50 to 500 mg. Time between start of treatment and recovery was recorded for 20 cats, and ranged from 1 to 10 days, with a mean of 4.4 days.

An antibiotic-treated cat that died had been treated with amoxicillin; treatment for the euthanatized cats included amoxicillin or ampicillin (2 cats), ampicillin and penicillin (1 cat), tetracycline (2 cats), and trimethoprim-sulfadiazine (1 cat). For those cats treated with a single type of antibiotic, the recovery rate was 95% for the tetracyclines. Other antibiotics were used solely for treatment too infrequently to interpret recovery rates accurately.

Clinicopathologic findings—Details regarding confirmatory laboratory test results were still available for 115 cases. Three early cases had been recorded as laboratory-confirmed, but the details on the tests are no longer available. A household with 2 cats with similar signs and onset times only had confirmatory laboratory testing done on 1 of the cats, and the second one was also reported as a case.

Thirty-nine percent (47 of 119) of the cases were confirmed with FA testing alone. Eighteen percent (21 cases) were confirmed with a single titer ≥ 32 after typical plague signs. Culture and FA results were positive for 11% (13) of the cats. Combinations of positive and negative test results were available for 29% (34) of the 119 cases.

Through FA testing or culturing, the *Y pestis* organism was identified in buboes in 42 cases and in blood in 9 cases. Other sites included abscesses (10 cases), spleen (8), lung (7), liver (7), wounds (1), and tongue lesion exudate (1).

Titers were reported for 50 cats, although acute and convalescent titers were available for only 14 of them. The number of days after onset of signs could be estimated for 50 of the serum specimens. The titers ranged from < 1:4 for a serum specimen taken 1 day after reported onset of signs, to 1:8,192 for a specimen taken 1 to 28 days after onset of signs. Follow-up titers were available for 8 cases. At 6 months after illness, 2 cats had titers of 1:16 and 1:32. At 9 months, the titers for 3 cats ranged from 1:256 to 1:2,048. At 1 year, the titers for 3 cats ranged from 1:1,024 to 1:8,192.

Information on WBC counts (WBC/μl) and differentials was available for only 14 cats (Table 3). The WBC counts were lower for more seriously ill cats, as defined by type of plague. A relative neutrophilia was reported for 7 of 11 cats, with a range of 80 to 100% segmented cells. An absolute neutrophilia was reported for 3 of 9 cases, with ranges of 12,600 to 25,344 neutrophils/μl. An increase in band cells was reported for 8 cats, with a range of 640 to 1,989 cells/μl (5 to 13%). Absolute lymphocyte counts were low for 7 cats (450 to 1,260 lymphocytes/μl), and 2 other cats had a relative lymphopenia (11 to 17%). Monocyte concentrations were normal for 9 cats, with reported values of 0 to 576 monocytes/μl (0 to 4%). Eosinophils were also normal for 9 cats, with counts of 0 to 576

Table 3—Reported WBC and differential counts (WBC/μl; n = 14)

Type of plague	Survival status	WBC	Segmented cells	Band cells	Lympho-cytes	Mono-cytes	Eosino-phils
Bubonic	S	28,800	88%	5%	3%	2%	2%
	E	16,000	NIR	NIR	NIR	NIR	NIR
	S	16,000	72%	5%	17%	NIR	4%
	S	15,300	81%	13%	5%	1%	0%
	S	14,000	90%	NIR	9%	1%	*
	S	10,800	74%	14%	9%	2%	1%
	E	NIR	69%	6%	19%	4%	2%
	E	NIR	"neutro-philia"	NIR	NIR	NIR	NIR
Septicemic/ Unclassified	S	14,100	11,280	987	1592	141	NIR
	S	9,700	NIR	"left shift"	NIR	NIR	NIR
	S	9,000	89%	8%	5%	1%	0%
	S	4,200	"all"	0	NIR	NIR	NIR
Pneumonic	S	4,700	62%	2%	25%	NIR	11%
	E	1,320	NIR	NIR	NIR	NIR	NIR

S = survived, E = euthanatized, NIR = no information reported.

eosinophils/μl (0 to 4%). Reported PCV values were normal for 4 cats (35 to 44%) and high for 1 cat (54%). Total proteins were normal for 1 cat (7.2) and high for 3 cats (8.4 to 8.9).

Pathologic findings—Necropsies were performed on 11 cats by local veterinarians or by pathologists at New Mexico Veterinary Diagnostic Services. Three of the cats necropsied were classified as having bubonic plague. Typical gross findings for these cats were emaciation, abscess formation (usually involving a lymph node), and abundant subcutaneous edema around the abscess. Microscopic examination of the abscesses revealed dense neutrophil infiltration. Fibrosis was present at the periphery of the abscesses, however, the abscesses were generally poorly encapsulated.

Four of the cats necropsied were classified as having pneumonic plague. The lung lesions tended to be focal, with each lesion being 1 to several centimeters in diameter. Microscopically, there was a dense infiltrate of neutrophils in alveoli and small bronchioles in the affected areas of lung (Fig 2). The borders of the lesions were indistinct. Numerous small coccobacilli were present in alveoli and bronchioles in the affected areas.

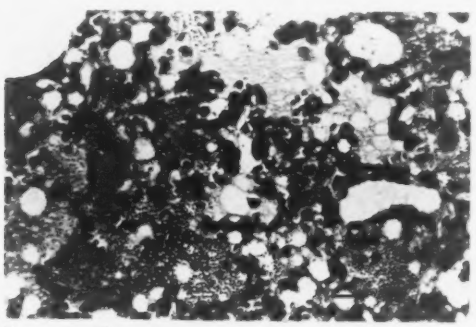

Figure 2—Photomicrograph of lung section from cat with pneumonic plague. Notice neutrophilic infiltrate and many gram negative bipolar staining coccobacilli, confirmed as Yersinia pestis. *Giemsa stain; bar = 20 μm.*

Four cats with neither buboes nor pneumonia (unclassified, presumed septicemic) were necropsied. Lesions were not found in 3 of these cats, 2 that had their signs confirmed as plague with a single high titer before euthanasia, and 1 that was confirmed as having plague by use of fluorescent antibody testing before it died. Interstitial pneumonia and focal necrotizing hepatitis were found in a fourth cat that had died after confirmation of plague infection with fluorescent antibody. However, *Y pestis* organisms were not isolated on necropsy. *Toxoplasma* organisms were present in both the lung and liver lesions, and it was thought that *Toxoplasma*, rather than *Y pestis*, was responsible for those lesions. Use of any antibiotic treatment was not reported for these 4 cases.

Discussion

Because most of the cats with plague in New Mexico had buboes, lethargy, anorexia, and fever, any cat with these signs and that lives in a plague-endemic area or has traveled from such an area should have appropriate plague diagnostic testing, particularly if it is a hunter.[14] In addition, veterinary staff must take care to reduce their own chances of infections through bites, open wound contacts with draining infective material, or inhalation of respiratory aerosols.

Because we have no surveys of the feline population of New Mexico with which to compare the observed signalment of the plague cases, we cannot definitively determine from this study whether age, gender, neutering status, or breed are risk factors for infection. Thus, these factors cannot be used at this time to rule in or rule out cases, but if these factors influence roaming and hunting, they might be expected to be correlated with risk of infection.

Most plague-related buboes will be submandibular or cervical and bilateral. However, lymphadenopathy can develop in other nodes, and in other nodes it will usually be unilateral. We believe that submandibular or cervical nodes become infected after oral inoculation of the organism through ingestion of infected rodents. Other lymph nodes probably enlarge after a flea bite in the area drained by the node. Although some of the buboes were abscessed at the time of diagnosis, most were not draining prior to veterinary intervention.

Cats with abscesses, lethargy, anorexia, and fever could have plague or cat bite abscesses, and a history of fighting with other cats or visible puncture wounds may help in the differential diagnosis.

Hunting cats from plague-endemic areas and that have pneumonia require the quickest response, because plague-infected aerosol could result in a large number of human primary pneumonic infections. In cats and human beings, pneumonic infections are difficult to treat successfully. Fortunately, pneumonic cases were only a small fraction of plague cases in cats.

Plague cases in cats with neither buboes nor pneumonia are probably missed most often, because the signs of fever, lethargy, and anorexia are consistent with many viral or bacterial infections. Cats infected with septicemic plague may have oral or gingival ulcerations, dyspnea, coughing, and sneezing similar to those attributable to the common calici and herpes viruses. It is not understood why people or cats with septicemic plague never develop palpable buboes.

Tularemia (*Francisella tularensis*) must also be differentiated from plague in cats with hunting histories. Both infections elicit a severe febrile reaction, and can become septicemic or pneumonic.[18] The geographic distribution of the 2 diseases overlaps (although tularemia is more widespread in the United States).[18] In the western United States, both diseases are more frequent in the warmer months.[19] However, few feline tularemia cases have been reported in New Mexico, so plague remains a more likely cause of clinical signs common to both diseases in this area.

Even in New Mexico, cats with these signs more often have diseases other than plague or tularemia. For example, of 117 FA tests completed for feline plague by our Scientific Laboratory Division in 1988, only 18 were positive for plague, and none were positive for tularemia. Of 137 cultures done, only 8 were plague-positive, and of 82 serologic analyses done, only 3 yielded a fourfold increase in titer, and only 2 were high single titers.

A suspect case of feline plague can be confirmed quickly with laboratory testing. Veterinarians can look for bipolar-staining, gram-negative coccobacilli on lymph node aspirates with Wayson or Giemsa stain,[10] but this is merely suggestive evidence. Lymph node or abscess aspirates should be tested with FA staining, the most sensitive and specific rapid diagnostic test.

Our data indicates that FA test results can be negative (possibly because of missing the organisms in the lymph node tap) and thus culturing and serologic analysis should also be considered for suspect cases. Organisms may be cultured from lymph node or abscess aspirates, tracheal washes, and blood, but results may not be available for up to 4 days. Septicemic plague is particularly difficult to define and confirm, and culturing of blood should be done.

A maximal increase in titers of 1:2,048 by day 12 has been observed in cats experimentally infected with *Y pestis*.[20] For naturally acquired infections in New Mexico, titers varied considerably even within 1 day of reported onset of signs. Owners of cats with plague frequently report their cats disappearing for several days before returning ill, so estimates of date of onset of signs are subject to error. However, on the basis of our data, cats are capable of generating an intense immune response to plague infection within days of exposure. Although it is difficult to determine time of exposure for naturally acquired plague in cats, cats with experimentally induced infections became ill within 24 to 48 hours of exposure.[20]

The duration of high antibody titer to the *Y pestis* antigen in naturally infected cats is unknown. In New Mexico cases, titers for some cats were low months after infection, whereas for others, the titers were as high as 1:8,192 a year after infection. These high titers could be related to subclinical reinfection in cats that continue to hunt and become exposed. In an experimental trial, by day 30, antibody titers decreased to a steady plateau of 1:512, which persisted through day 300.[20]

When a cat has signs suggestive of plague, a single titer $\geq$1:32 is usually considered diagnostic, but could indicate a previous rather than current infection. To our knowledge, serosurveys of nonill cats have not yet been done in New Mexico to ascertain the frequency of infection. A single titer lower than 1:32 taken during the first several days of clinical signs may not rule out plague because the immune system in some cats has not yet had time to respond. A fourfold increase in titers taken at least 2 weeks apart confirms concurrent plague infection.

An absolute neutrophilia with a left shift has been observed by other authors in cats with plague.[11] The WBC counts and differential counts were not reported for most of New Mexico's plague cases in cats. For those reporting, a relative neutrophilia with a left shift and lymphopenia was most frequently observed.

The lesions found in the 11 cats necropsied were similar to those previously described in experimentally infected[20] and naturally infected[11] cats. In the bubonic and pneumonic cases, the lesions tended to be focal, suppurative, and poorly defined. Numerous small bacteria could generally be found in the lesions. Cats with the septicemic form of plague typically had no gross or microscopic lesions that could be attributed to the plague organism.

Because of high mortality, potential cat-to-human being and cat-to-cat transmission, cats suspected of having plague are usually treated with antibiotics presumptively until laboratory confirmation is available. Studies of antibiotic effectiveness against plague organisms have been performed primarily in vitro and in human beings, not in cats. Thus we have recommended and New Mexico veterinarians have used the same antibiotics for feline cases as those recommended for human cases.

Although the higher survival rate in those cats with plague that are treated with antibiotics offers support for a causal association between antibiotic treatment and recovery, an alternative explanation is the opposite causal association, that is, those cats more likely to recover (such as those with bubonic plague) were more likely to be given antibiotics. On the basis of data for the New Mexico cases, both

phenomena were occurring. However, even in the cats with more serious signs, there was a higher survival rate with antibiotic treatment, as indicated by the fact that all of the cats with pneumonic plague and that were treated with antibiotics survived.

Tetracyclines appear to be the most effective antiobiotics in uncomplicated cases and for plague-infected cats without clinical signs.[8] We have recommended oral administration of tetracycline to cats with such cases at a rate of 25 mg/kg of body weight, q 8 h.[21] In human cases, chloramphenicol and aminoglycosides such as streptomycin and gentamicin are believed to be more effective in severe infections,[8] and were used successfully in some of the cats in New Mexico. Trimethoprim-sulfamethoxazole is used frequently to treat children with plague, and was also used successfully in a few cats with plague in New Mexico. Antibiotics in the penicillin and ampicillin families have not been shown to be efficacious in vitro, and our observed lower recovery rate with ampicillin supports this. The 3 cats treated with penicillin alone survived; however, this is a small number of cats and they probably would have recovered without antibiotic treatment.

It is not known whether the recent increase in plague cases in cats in New Mexico is a result of higher incidence of infection, improved surveillance, or increased awareness on the part of veterinarians. The incidence of plague in human beings appears to follow a 5-year cycle, possibly related to epizootics in host rodents with high mortality.[4] The seasonal distribution of plague in cats and human beings reflects the reduced activity in the winter of rodents, rodent fleas, human beings, and their pets. Dog and cat fleas do not appear to be involved in plague transmission, and are uncommon in plague endemic areas in New Mexico.

Prevention of plague infection is important because diagnosis in time for effective treatment of plague-infected cats and human contacts is often difficult. To reduce the risk of being infected with plague and transporting plague-infected fleas into the home environment, owners who live in plague-endemic areas should consider dusting their cats with flea powder weekly (especially during the summer months), preventing their cats from hunting, and eliminating rodent harborage such as wood piles, junk piles, or garbage.

Veterinarians and their staffs can reduce their risk of acquiring plague by treating cats for fleas as soon as plague is suspected, isolating cats with suspect plague to prevent human and cat contact with draining abscesses and pneumonic aerosols, and using mask and gloves when handling live or dead cats suspected to have plague. Specimens should be submitted as soon as possible to a qualified laboratory to have plague confirmed, so that contacts can be evaluated for antibiotic prophylaxis.

Additional studies are necessary to investigate the geographic and temporal risks of acquiring plague infections. Serologic studies in dogs[22-24] are useful because their infection is usually subclinical and self-limiting.[8,20,24,25] In addition, there should be further investigation with experimental studies of the feline subclinical and case fatality rates. Better documentation of septicemic plague in cats is needed by submission of blood for cultures, and more deceased/euthanatized cats should be submitted for necropsy and histologic analyses. Finally, better definition of potential risk factors would be useful, including lack of neutering, rodent and flea exposure a week before illness onset, and lack of flea control.

[a]Ted Brown, Vector Control Program, New Mexico Environment Department, Santa Fe, NM. Personal communication, 1990.

References

1. Simpson WJ. *A treatise on plague*. Cambridge, England: Cambridge University Press, 1905;22,104–115, 223.
2. Isaacson M, Levy D, Pienaar BJ, et al. Unusual cases of human plague in southern Africa. *S Afr Med J* 1973;10:2109–2113.
3. Thornton DJ, Tustin RC, Pienaar BJ, et al. Cat bite transmission of Yersinia pestis infection to man. *J S Afr Vet Assoc* 1975;46:165–169.
4. Green W, Gorman G, Counts JM, et al. Plague—Arizona, Colorado, New Mexico. *Morbid Mortal Weekly Rep* 1977;26:213.
5. Heaton E, Gaskin H, Marchiando K, et al. Plague—United States. *Morbid Mortal Weekly Rep* 1977;26:337.
6. Fitch R, Smith L, Christenson SL, et al. Feline plague—California. *MMWR* 1977;26:362.
7. Center for Disease Control, Vector-Borne diseases division. *1979 report. Part II. Plague activities*. Fort Collins, Colo: Centers for Disease Control, 1979;14–59.
8. Poland J, Barnes AL. Plague. In: Steele JH, ed. *CRC handbook series in zoonoses. Section A: bacterial, rickettsial, and mycotic diseases*. Boca Raton, Fla: CRC Press, Inc, 1979;515–556.
9. Human plague case—Jefferson County. *Colorado disease bulletin* 1981;9:1.
10. Kaufmann AF, Mann JM, Gardiner TM, et al. Public health implications of plague in domestic cats. *J Am Vet Med Assoc* 1981;179:875–878.
11. Rollag OJ, Skeels MR, Nims LJ, et al. Feline plague in New Mexico: report of five cases. *J Am Vet Med Assoc* 1981; 179:1381–1383.
12. Wenger BG, Warren AJ, Forseth V, et al. Human bubonic plague transmitted by a domestic cat scratch. *JAMA* 1984;251:927–928.
13. Werner SB, Weidmer CE, Nelson BC, et al. Primary plague pneumonia contracted from a domestic cat at South Lake Tahoe, Calif. *JAMA* 1984;251:929–931.
14. Eidson M, Tierney LA, Rollag OJ, et al. Feline plague in New Mexico: risk factors and transmission to humans. *Am J Pub Health* 1988;78:1333–1335.
15. Plague pneumonia—California. *MMWR* 1984;33: 481–482.
16. Meyer KF. The ecology of plague. *Medicine* (Baltimore) 1942;21:143–174.
17. Meyer KF. Plague. *Med Clin North Am* 1943;27:745–765.
18. Siegmund OH, ed. *The Merck veterinary manual*. Rahway, NJ: Merck & Co, Inc, 1979;412–415.

19. Brooks GF, Buchanan TM. Tularemia in the United States: epidemologic aspects in the 1960s and follow-up of the outbreak of Tularemia in Vermont. *J Infect Dis* 1970;121:357–359.

20. Rust JH, Cavanaugh DC, O'Shita R, et al. The role of domestic animals in the epidemiology of plague. I. Experimental infection of dogs and cats. *J Infect Dis* 1971;124:522–526.

21. Pharmacy Committee, Colorado State University Veterinary Teaching Hospital. *Formulary* 1982–1983.

22. Rust JH, Miller BDM, Bahmanyar M, et al. The role of domestic animals in the epidemiology of plague. II. Antibody to Yersinia pestis in sera of dogs and cats. *J Infect Dis* 1971; 124:527–531.

23. Shepherd AJ, Leman PA, Hummitach DE, et al. A comparison of serological techniques for plague surveillance. *Trans R Soc Trop Med Hyg* 1984;78:771–773.

24. Archibald WS, Kunita sJ. Detection of plague by testing serums of dogs on the Navajo reservation. *HSMHA Health Reports* 1971;86:377–380.

25. Gregg CT. *Plague*. Albuquerque, NM: University of New Mexico Press, 1985;282–297.

Book Review: Swine Nutrition Guide

This paperback is a useful, inexpensive introduction to applied swine nutrition. The text is easily understood and includes reliable information about criteria that influence feedstuff selection and diet composition. Special attention has been paid to the use of major Western feedstuffs in swine nutrition.

Two very informative chapters cover the nutrient requirements of pigs and discuss the potential use of a wide variety of feedstuffs in swine rations. Four other chapters address feeding regimens and diet composition for pigs in various production stages. The text includes tables with sample diets for each production group and discusses the various factors that affect feed intake and use. Other sections cover methods of diet formulation, feed processing techniques, and toxin contamination of feed.

This text will be a valuable source of information for students, veterinary practioners, and managers of swine operations who wish to acquire a basic understanding of swine nutrition. Selected references at the end of each chapter direct the reader to more specific and detailed information. The text lacks concise, complete summaries in regard to the intended audience. Several chapters present information in discussion form; other sections highlight important information in numerous small windows. The use of the text for self-instruction or as a quick reference is hampered by this approach. The authors should consider the introduction of summary charts that group and contrast essential information of whole chapters or related topics. —*[Swine Nutrition Guide. By John Patience and Phil Thacker. 260 pages; illustrated. Saskatchewan, Saskatoon. Canada S7N OWO 1989. Price $21.50; in USA $22.00.]*—ANTON L. PRESSING

NOTE

ONLY THE FOLLOWING PAGES OF THIS

PUBLICATION HAVE BEEN FILMED

Cov. 1, Pgs: 1234-1236,

1348-1353, 1365-1373